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Pulmonary artery and left heart end-diastolic pressure relations[1]

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The exponential fall of the pulmonary pressure curve from its systolic peak to left atrial pressure previously described in the presence of normal intraventricular conduction has been investigated in patients with right bundle-branch block. We have been able to show once more that the pulmonary artery diastolic pressure seeks the left atrial level: when right ventricular systole is delayed the pulmonary artery pressure continues to fall with the left atrial 'x' descent.

The absence of an end-diastolic gradient across the *normal* pulmonary vascular bed has been reported (Kaltman *et al.*, 1966; Rapaport and Scheinman, 1969) and related to the low pulmonary vascular resistance which permits the rapid equilibration of pressure across the 'bed' by end-diastole.

The pulmonary artery pressure decays in an exponential fashion (Shaw, 1963) from its peak to the level of the left atrial pressure – its ultimate outflow resistance (Hamilton, Woodbury, and Vogt, 1939). This equilibration is interrupted by the next pulse opening the pulmonary valve.

If all the above observations are valid so that the pulmonary artery pressure rapidly seeks the left atrial level as reported (Hamilton *et al.*, 1939; Kaltman *et al.*, 1966; Rapaport and Scheinman, 1969), then a condition in which right ventricular systole is delayed should permit the pulmonary artery pressure to continue to fall with the 'x' descent of the left atrium, thus resulting in a pulmonary artery end-diastolic pressure lower than the left ventricular end-diastolic pressure. The following study shows that this hypothesis is correct as such dynamics do occur in the presence of either complete right bundle-branch block or a right-sided conduction disturbance.

Methods

Selection of patients The records of the last 500 patients studied in this laboratory were reviewed. Six patients with a right-sided conduction disturbance had had simultaneous right and left heart catheterization and were therefore selected for this study. One additional patient in whom a transient right-sided conduction disturbance was produced during catheterization is also included (Case 7) (Table).

Though a number of additional patients with a similar electrocardiographic abnormality also showed the unusual pressure relations that are the substance of this report, only those in whom simultaneous pressures were recorded have been included in the study.

Also excluded were right bundle-branch block patients with pulmonary vascular disease, and those with tight pulmonary stenosis in whom the contours of the diminutive pulmonary artery pressure trace were too distorted for interpretation.

Technique All patients underwent simultaneous right and left heart catheterization in the usual manner. The pulmonary artery pressures were recorded through a 125 cm. no. 6 Cournand catheter.[1] The left ventricular pressures were recorded through a 100 cm. no. 8 Shirey ventriculography catheter.[1]

Pressures were monitored with Statham P23db

[1] U.S.C.I., Glen Falls, New York.

TABLE *Haemodynamic and electrocardiographic data on seven patients*

Case	*Age*	*Sex*	*Diagnosis*	*QRS duration (sec.)*	*Pressure (mm. Hg)*	
					Pulmonary artery	*Left ventricle*
1	13	M	Ventric. septal defect	0·12	25/12	94/15
2	19	M	Pulm. stenosis	0·11	20/9	116/12
3	61	M	Arteriosclerotic heart disease	0·11	28/9	160/16
4	17	M	Atrial septal defect	0·13	20/9	113/11
5	20	M	Pulm. stenosis	0·10	20/13	116/15
6	16	M	Atrial septal defect	0·11	29/7	125/13
7	16	F	No heart disease	0·11	24/6	104/10

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gauges on a 12-channel Electronics for Medicine[2] recorder. All gauges were levelled via a common fluid-filled manifold to a point 5 cm. below the angle of Louis. The gauges were then brought to equal sensitivity by imposing several known pressures into the manifold, and adjusting the fine sensitivity control until all gauges read true and equal pressures. Recordings were made with both low and high gain amplification. The averages of the pressures measured over two respiratory cycles were reported.

The paired catheter system was tested to show its pressure transmission characteristics in the following manner. An open top glass test chamber was tightly fitted with a two-holed rubber stopper at the bottom. The two catheters were threaded through the holes into the chamber which was then filled with enough saline to cover the catheters. A 3 in. no. 20 needle was then forced through the stopper and advanced until the point was above the fluid level. The hub was connected to a hand-bulb mercury manometer. The catheters were then attached to their respective gauges and brought to equal sensitivity as described above. The open top of the test chamber was covered with a rubber membrane and pressure was introduced through the needle. While recording with a paper speed of 100 mm. a second, the membrane was ruptured by lightly touching it with a hot soldering iron. The response times of the paired catheters were then compared (Fig. 1).

[2] Electronics for Medicine, White Plains, New York.

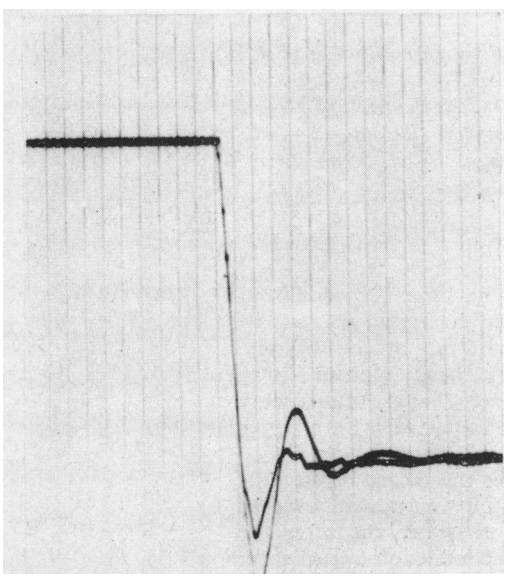

FIG. 1 *The response times of a paired 100 cm. and 125 cm. catheter each connected to a Statham P23db gauge are seen to be almost identical. Paper speed 100 mm./sec. Time lines 0·04 sec.*

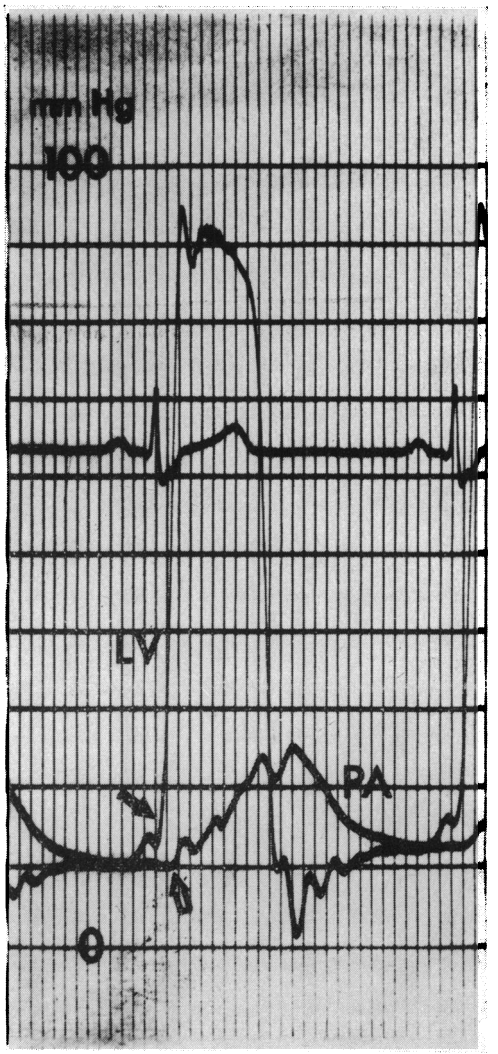

FIG. 2 *Simultaneous pulmonary artery (PA) and left ventricular (LV) pressures recorded from Case 1. The solid arrow indicates left ventricular end-diastolic pressure and the open arrow the end-diastolic pressure in the pulmonary artery. The electrocardiogram shows the right bundle-branch block pattern. The horizontal lines represent increments of 10 mm. Hg from 0 to 100 mm. Hg. Time lines 0·04 sec. Paper speed 50 mm./sec.*

Results

The temporal relations of the pressure transmission of the paired catheter system are seen in Fig. 1. Paper speed is 100 mm./sec. and the time lines are 0·04 sec. The response times are very nearly identical and are consistent with the studies of Laurens (1966).

The pertinent electrocardiographic and haemodynamic data appear in the Table.

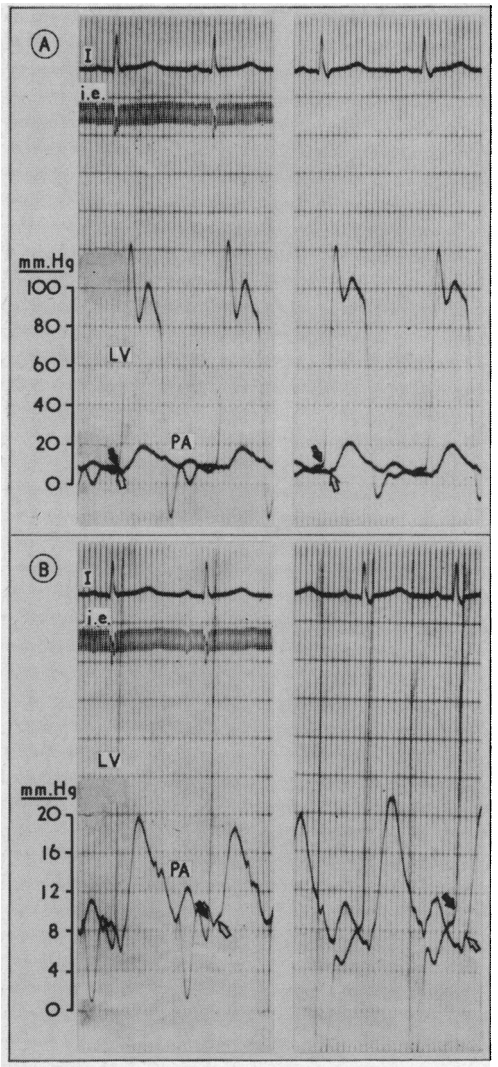

FIG. 3 *(A) A low gain amplification of the simultaneous left ventricular (LV) pulmonary artery (PA) pressure relations before and after the onset of a right-sided conduction disturbance. The solid arrow indicates the left ventricular end-diastolic pressure and the open arrow the end-diastolic pressure in the pulmonary artery. The electrocardiogram is standard lead I. The development of a wide S wave is seen, i.e.= internal electrogram (recorded in the pulmonary artery). Time lines 0·04 sec. Paper speed 50 mm./sec. (B) A high gain amplification of the same sequence.*

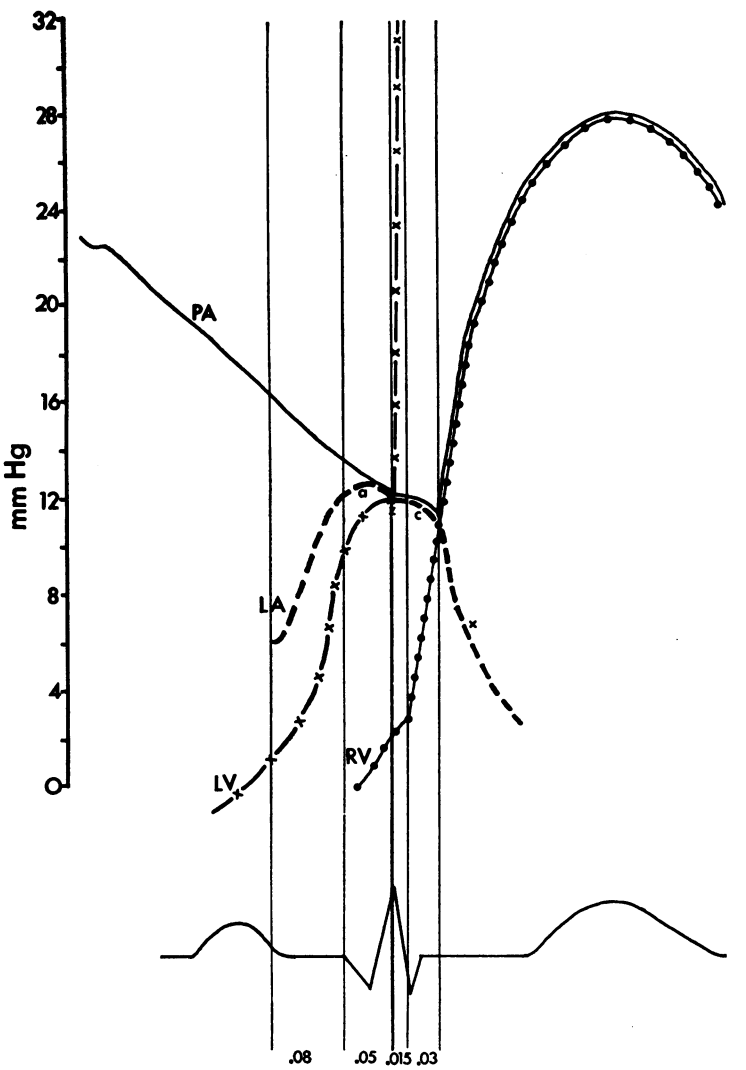

FIG. 4 *The time and pressure relations of the pulmonary artery (PA————), left atrium (LA– – – – –), left ventricle (LV—x—x—), and right ventricle (RV ●—●—●—●). The components of the left atrial curve are labelled in the usual manner ('a', 'z', 'c', and 'x'). See text.*

In every instance, the simultaneously recorded left ventricular end-diastolic pressure was higher than the pulmonary artery end-diastolic pressure (range 2–7 mm. Hg). The QRS durations ranged from 0·10 to 0·13 sec.

The simultaneous pulmonary artery and left ventricular pressure curves recorded from Case 1 are reproduced in Fig. 2. The arrows indicate the different levels of end-diastolic pressure in each with the pulmonary artery end-diastolic pressure being measurably lower. Time lines are 0·04 sec. with a paper speed of 50 mm./second.

Panel A of Fig. 3 is the record of the pulmonary artery and left ventricular trace at low gain amplification before and after the onset of a right-sided conduction disturbance.

Panel B displays the same sequence at high gain amplification.

Discussion

The normal pulmonary vascular bed permits a pressure equilibration by end-diastole (Hamilton *et al.*, 1939; Kaltman *et al.*, 1966; Rapaport and Scheinman, 1969). The right bundle-branch block patients had no disease of the pulmonary vascular bed and had pulmonary artery end-diastolic pressures consistently lower than those simultaneously observed in the left ventricle. The following explanation predicated upon atrioventricular temporal relations seems to account for the observations made.

Fig. 4 is a drawing depicting the classic relations of the pressures in the pulmonary artery, left atrium, left ventricle, and right ventricle. The intervals are those reported by Braunwald, Fishman, and Cournand (1956). The left ventricular end-diastolic pressure which corresponds to the left atrial 'z' point occurs approximately 0·05 sec. after the Q wave onset (Braunwald *et al.*, 1956). Right ventricular systole occurs 0·015 sec. later (Braunwald *et al.*, 1956). The right ventricular ejection rate (with a dp/dt average of 259 mm. Hg/sec.) as reported by Gleason and Braunwald (1962) would cause the pulmonary artery semilunar valves to open 0·10 sec. after the Q wave. The calculation is based on a right ventricular end-diastolic pressure of 3 mm. Hg and the preceding pulmonary artery end-diastolic pressure of 12 mm. Hg.

Right bundle-branch block has two basic effects on right ventricular contraction. The first is to exaggerate the normal ventricular asynchrony, resulting in a further delay of right ventricular systole (Braunwald *et al.*, 1956; Erickson, Scher, and Becker, 1957; Luisada, 1962; Johnston *et al.*, 1966; Beller, 1967; Herbert, 1969). The second factor which would also delay the opening of the pulmonary valve is the reduction in dp/dt. The reduced rate of the right ventricular pressure rise is attributed to the loss of synchronous contraction of the right ventricular muscle fibres due to the aberrant conduction. Johnston *et al.* (1966) reported a 41 mm. Hg/sec. reduction in the rate of the right ventricular pressure rise. Applying their results (Braunwald *et al.*, 1956; Erickson *et al.*, 1957; Luisada, 1962; Johnston *et al.*, 1966; Beller, 1967; Herbert, 1969), the aggregate delay in the pulmonary valve opening would be 0·02–0·03 sec. beyond normal. This delay occurs during the period of the initiation of the rapid left atrial 'x' descent. Fig. 5 shows these relations. In the presence of a normal left

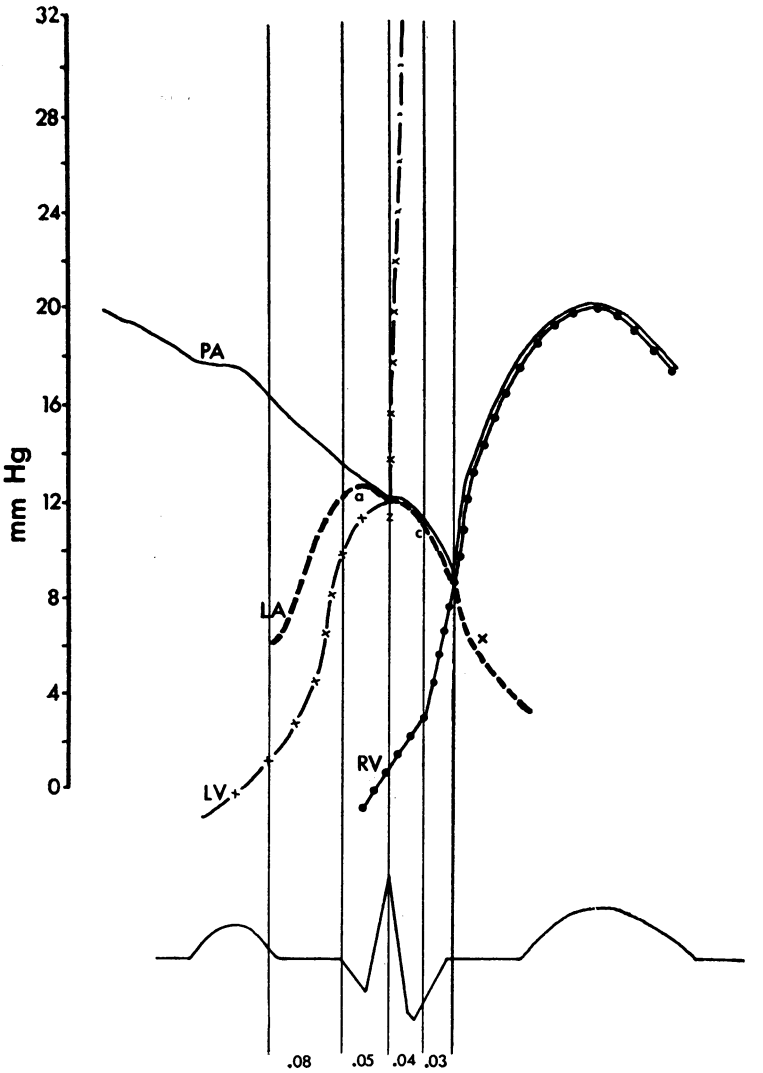

FIG. 5 *The effect of right bundle-branch block on pulmonary artery, left atrial, left ventricular, and right ventricular pressure relations. All abbreviations as in Fig. 4.*

atrial and left ventricular time sequence, the pulmonary artery pressure curve continues to follow that in the left atrium for another 0·03 sec. During this time, the pressure falls 3 mm. Hg before interruption by the next right ventricular pressure pulse.

The finding that a right bundle-branch block permits the pulmonary artery end-diastolic pressure to fall below that in the left ventricle is further evidence for the observation that there is little friction in the normal pulmonary vascular bed (Hamilton *et al.*, 1939; Doyle *et al.*, 1957; Kaltman *et al.*, 1966; Rapaport and Scheinman, 1969). No attempt has been made to evaluate these

dynamics in terms of the overall function of the heart. One must note, however, that the fall of the pulmonary artery end-diastolic pressure to lower levels means that the right ventricle will eject against a lower pressure and this may perhaps compensate for the reduction in dp/dt. In fact the time lapse from right ventricular systole to ejection is the same with right bundle-branch block as it is during normal conduction. Though dp/dt is less, the delay in systole permitting the pulmonary artery end-diastolic pressure to fall further requires less of a pressure rise in the right ventricle before it is able to open the pulmonary valve.

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